## The value of different methods and models: comment on Slomkowski et al

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# Commentaries

## BROTHER, CAN YOU SPARE A SMOKE? SIBLING TRANSMISSION OF TOBACCO USE

There has been much recent excitement concerning the apparent heritability of tobacco smoking and the identification of candidate genes that might contribute to this heritability (Tyndale 2003). Although the genetic contributions to smoking behavior hold significant explanatory potential, sometimes lost in the excitement is that we have yet to reach a satisfactory understanding of the environmental contributors to this behavior. Thus, it is particularly encouraging when a behavioral genetic design is used to isolate environmental effects by controlling for genetic effects, as Slomkowski *et al*. (2005) accomplished. This strategy has been underused in general, and particularly so within the tobacco literature. Yet the power of the design allowed Slomkowski *et al*. to demonstrate that the nature of sibling relationships (i.e. social connectedness) is associated with the transmission of smoking between the siblings.

One immediate question that emerges from these findings is how smoking behavior is transmitted between siblings via their relationship. That is, what are the units of communication? We suggest that the answer is likely to be found among the individuals' expectancies regarding cigarette smoking (Brandon *et al*. 1999). Expectancies are the memory templates (both implicit and explicit) of the reinforcing value of a substance that can influence substance use (Goldman 1999). They are predictive of all phases of substance onset, maintenance and cessation, and they may serve as the final common pathway of multiple determinants of substance use and abuse. Thus, expectancies about the immediate and delayed, positive and negative consequences of smoking are natural candidates for investigation as the units of transmission between siblings.

It is impressive that the findings of the study emerged despite the use of an admittedly crude tobacco use measure—smoking frequency. The identification of smoking-related endophenotypes (an endogenous characteristic of a person that is a more direct product of a genotype; Iacono 1998), such as physiological reactivity to nicotine, might improve the partitioning of genetic variance, control for it more completely, and therefore elucidate environmental effects. Additionally, the dependent measure, while an improvement over a dichotomous smoker/non-smoker variable, none the less fails to capture the variability in either smoking behavior or tobacco dependence. This has been a limitation of much tobacco research, but there has been recent progress in developing sensitive, multi-modal indices of tobacco dependence (e.g. Piper *et al*. 2004). Indeed, a recent issue of this journal was dedicated to theory-based indices of tobacco dependence among adolescents p*er* se (see Brandon *et al*. 2004; Eissenberg 2004; Glautier 2004).

In sum, by identifying an apparently robust environmental influence on adolescent smoking, Slomkowski *et al*. 2005) have set the stage for future research that can distill this effect by examining the units of sibling transmission and refining the measurement of tobacco use and dependence. Although substantial environmental variance in smoking remains to be identified, genetically informative designs offer the potential for further progress in this regard, through the isolation first of genetic influences, and now sibling transmission.

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**Table 1** Estimation of genetic and shared environmental influences on maximum number of cigarettes smoked per day in total group and separately for twin pairs who have daily/weekly contact and twin pairs who contact each other less than weekly. Genetic and shared environmental influences are estimated using the DeFries-Fulker regression model (DeFries & Fulker 1985). Regression coefficient $b3 = h^2$ and regression coefficient $b1 = c^2$. Sample: twins participating in the survey of 2000 and/or the survey of 2002 of the Netherlands Twin Registry, mean age: 32 years.

| | $b_3$ ($h^2$) | $b_1$ ($c^2$) |
|---|---|---|
| Total group | 0.54 ($P = 0.000$) | 0.15 ($P = 0.032$) |
| Twin pairs: daily/weekly contact | 0.33 ($P = 0.018$) | 0.38 ($P = 0.001$) |
| Twin pairs: less than weekly/no contact | 0.61 ($P = 0.000$) | 0.06 ($P = 0.510$) |

## THE VALUE OF DIFFERENT METHODS AND MODELS: COMMENT ON SLOMKOWSKI *ET AL.* (2005)

Slomkowski *et al.* (2005) have used the DeFries-Fulker regression model to test whether sibling effects on smoking may reflect social or genetic processes. They included not only the siblings phenotype but also the social connectedness between the siblings and showed that shared environmental sibling effects on smoking were significant.

The authors used the measure 'number of days smoked over the past 30 days'. Although the authors do not report the distribution of this variable, it is probably not normally distributed. It is expected that a large group will score 0 days (non-smokers) and a group will score 30 days (daily smokers). Probably a small group will report a number between 1 and 29 (chippers, non-daily smokers). The authors examined the individual differences on this variable and transformed it to approximately a normal distribution. This measure is especially useful in the critical period of taking up smoking. After smoking is initiated other measures, such as number of cigarettes per day, number of quit attempts, years smoked and nicotine dependence, become more important. It is interesting whether the results of Slomkowski *et al.* can be replicated for these types of measure. In a sample of Dutch twins and their family members data on smoking and social contact were collected (Boomsma *et al.* 2002). For the maximum number of cigarettes smoked per day, the environmental effects are stronger in twins who have contact on a daily/weekly basis than twins who contact each other less than weekly (Table 1).

Both the results of Slomkowski *et al.* and the results of our Dutch data are in agreement with previous studies. Rose *et al.* (1990) concluded that social contact contributed to sibling resemblance for alcohol consumption. Slomkowski *et al.* state that the critical addition of their report is the demonstration that sibling social connectedness is a significant moderator of the shared environmental effect on smoking adolescence using a genetically informative design. However, Rose *et al.* (1990) have already introduced a twin-model including social contact. They considered and answered the question of what the direction of the effect is: does similarity in alcohol consumption lead to increased contact or does more contact lead to similarity in alcohol consumption? They concluded that more social contact contributed to higher sibling similarity in alcohol consumption (Rose *et al.* 1990). Several important smoking measures are dichotomous, such as current smoking (yes/no) or ever smoked (yes/no). Carey (1992) has introduced a method of analysis that permit reciprocal twin interactions for a dichotomous trait. This model provided a better fit to their data (on antisocial behaviour) and yielded lower estimates of heritability compared to the traditional twin-model. Those threshold models including sibling interaction could also be applied to dichotomous smoking data. Using different methods and models can shed light on the nature, timing and specificity of sibling effects on smoking behaviour.

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## SIBLING EFFECTS ON SMOKING IN ADOLESCENCE: EVIDENCE FOR SOCIAL INFLUENCE FROM A GENETICALLY INFORMATIVE DESIGN: COMMENT ON SLOMKOWSKI *ET AL.* 2005

This important report by Slomkowski *et al.* (2005) underscores the importance of genetically informed research designs for gaining new understanding of environmental influences on the use of tobacco, alcohol or other addictive substances. The paper accomplishes this goal in at least two ways. First, it demonstrates that the smoking behavior of one sibling is related significantly to the smoking behavior of a second sibling, even after controlling for the degree of genetic relatedness between the two siblings. Indeed, the findings showed that the correspondence in sibling smoking was equally high for unrelated as for full siblings or dizygotic twins in at least some situations. Results such as these make it clear that the shared environments of siblings, in addition to non-shared influences and genetic similarities, are likely to play a powerful role in generating risk for tobacco and, quite probably, other types of substance use. This demonstration of environmental effects net of common genes simply cannot be achieved in the absence of a genetically informed research design.

A second important contribution of the study was to show that a potentially important interpersonal mechanism, social connectedness, provided a plausible means through which siblings might influence one another's smoking behaviors. That is, the findings indicated that the shared environmental effect, but not the genetic effect, was moderated by social connectedness. When siblings were highly involved with one another in diverse activities, then one sibling's smoking was especially likely to be associated with the second sibling's smoking. Again, this influence operated net of any genetic effects. It seems reasonable that siblings who spend time together, and who share common friends and activities, would be more likely to engage in substance-using behaviors to a similar degree, if for no other reason than that their high level of social involvement would provide numerous opportunities for one sibling to be present when the other sibling is smoking, drinking or using illicit drugs. When common and perhaps substance-using friends are added to this equation, it seems it would be difficult for an initially non-smoking sibling not to at least experiment with smoking.

As important as these two major contributions are, however, the study also sets the stage for gaining more complete understanding of the multiple pathways through which family and peers probably affect risk for smoking and other forms of substance use. Simply put, genetically informed studies of this type increase confidence in the reality of environmental influences on variations in individual behaviors, and that confidence enhances the value of more traditional observational studies that do not include a behavioral genetics component. For example, Slomkowski and colleagues found that peers, parents and siblings had independent effects on the smoking behavior of a focal sibling. To be sure, additional research needs to be conducted to gain a more thorough understanding of sibling effects alone; however, there is also a need to understand the larger social environment in which sibling relationships operate.

To illustrate this point, over a decade ago my colleagues and I (Melby *et al.* 1993) examined the empirical credibility of a theoretical model that postulated specific connections among parental, peer, sibling and a focal adolescent's ($n = 204$) tobacco use. The results of that study showed that parent and sibling smoking were related to the acquisition of peers who also smoked by the focal adolescent. The findings indicated that parent and sibling smoking in a sense 'granted permission' to the focal adolescent to associate with tobacco-using peers, who had a direct influence on the focal adolescent's smoking. Interestingly, however, the child-rearing style of parents also affected association with peers who smoked, suggesting that the substance-using behaviors of parents were only part of the story regarding family influence. Other reports in this program of research have shown that parenting style moderates sibling influences on alcohol use and problems with alcohol (Conger *et al.* 1994) and that sibling effects may be indirect through peer associations in affecting change in drinking behaviors (Conger & Rueter 1996). These findings suggest that future research should look not only at the internal dynamics of sibling relationships to understand their influence, as proposed by Slomkowski and her collaborators, but also at the broader social context of sibling relationships and how it shapes and is shaped by the sibling bond. Such research will take full advantage of the important contributions of the current study.

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## THE SIGNIFICANCE OF SOCIAL CONNECTEDNESS: COMMENT ON SLOMKOWSKI *ET AL.* 2005

Despite the dramatic decrease in smoking during the last decade in the United States of America, nearly half of adolescents during the past five years have experimented with tobacco (Mowery *et al.* 2004). This underscores the urgent need for the development of effective preventive strategies in youth. The study by Slomkowski *et al.* (2005) advances our understanding of the risk factors for adolescent smoking behavior. Based on evidence of greater similarity between peers than siblings, the lack of a strong association between parental and offspring smoking, environmental similarity playing a greater role than genetic relatedness among twins, and comparable correlations between unrelated and related siblings, they conclude that social factors are far more potent than genetic and biologic factors in determining both smoking initiation and progression. The convergence of evidence regarding the critical importance of common environmental factors underlying adolescent smoking across numerous studies despite disparate samples, methods, and analyses (Koopmans *et al.* 1999; Tyas *et al.* 1998; McGue *et al.* 2000; Hopfer *et al.* 2003; Vink *et al.* 2003; White *et al.* 2003) strengthens the validity of the findings of the current study.

The unique contribution of this work is its actual identification of a potential mechanism for familial similarity; namely, that social connectedness moderates the association of smoking between siblings. Since one component of social connectedness is mutual friends, the potent effect of social influences suggests that peer smoking is not independent of sibling smoking. This establishes a more complex mechanism for social contextual influences on smoking than simply additive influences of social risk factors. Even though only one aspect of sibling interaction is included, this paper provides a model for the type of evidence that will be necessary to translate knowledge gleaned by twin and family studies into prevention. The prospective design, although only one year, also enables prediction of specific familial influences on the incidence and progression of smoking over time.

On a broader level, this study also illustrates the value of the application of existing data to address major public health problems. There is an abundance of similar data sets with valuable information that could be employed to advance our knowledge regarding the risk factors and consequences of health-related behaviors. Although there are few of this quality and magnitude to address the specific correlates of smoking in youth, papers reporting findings such as those in the present study should encourage other investigators to take advantage of existing resources, particularly to develop a priori hypotheses before embarking upon new studies.

There are several implications of this work that advance knowledge from prior research on influences on youth smoking. First, prevention efforts should focus on the contextual basis of adolescent smoking rather than on individual adolescents. Second, the risk factors for adolescent initiation should be distinguished from those for continuation of smoking. Third, the determinants of adolescent smoking may differ from those for older youth, as well as between males and females. Finally, the low attributable risk of genetic factors underlying adolescent smoking demonstrated in this and nearly all other studies of youth suggest that identification of genes will have little utility in reducing smoking. Therefore, the current investment of substantial effort to identify genes for smoking behavior is not well justified in adolescent samples. Even if genes conferring increased vulnerability to smoking were identified, it is not clear how this information could be translated into a realistic prevention program. By contrast, identification of environmental mechanisms for vulnerability would be likely to have far greater impact on the translation into primary prevention programs.

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## IMPROVING UNDERSTANDING OF SIBLING EFFECTS ON ADOLESCENT SMOKING: RESPONSE TO THE COMMENTARIES

We appreciated the thoughtful comments provided by the four commentaries on our paper (Slomkowski *et al.* 2005). The commentaries included a number of converging opinions as well as specific suggestions to improve our understanding of sibling influences on smoking. All of the commentators agreed with our position that genetically informative designs may be utilized to generate evidence of environmental influence on behavioral phenotypes, especially when theory-driven measures of putative environmental influence are utilized. Thus Merikangas' (2005) point that behavioral genetic designs may inform prevention strategies by yielding information on environmental mechanisms is well-taken, especially with reference to initiation of substance use in adolescence, which often shows evidence of shared environmental influences of equal or greater magnitude than genetic effects (Rende & Waldman, in press).

Vink (2005) provides a number of important conceptual and methodological considerations for such behavioral genetic work. One critical point is the measurement of the phenotype, an issue also addressed by Brandon & Brandon (2005). These authors emphasize both the complexity of smoking phenotypes and progress that is being made in the field in measuring a range of smoking indices, especially with adolescent populations. The data presented by Vink provide expansion of our findings by focusing on 'maximum number of cigarettes smoked in a day', a more specific indicator of adolescent smoking than the frequency measure used in our study. Vink also brings attention to the seminal work of Rose and colleagues (Rose *et al.* 1990), which is a good reminder that behavior genetics research has, for many years, generated important information on the social environment. As suggested by Vink, there are indeed a number of behavioral genetic models well-suited to address social influences on smoking, especially as they may be adapted to various representations of the smoking phenotype (e.g. categorical vs. continuous). We add to these good points the importance of broadening our conception and measurement of environmental influences in behavioral genetic models. For example, our paper expands the concept of social contact between siblings to include ratings of affection for each other and time spent with mutual friends, along with time spent together. Much of our current work emphasizes intensive methods for studying social interaction (e.g. micro social coding of videotaped interactions; recording sibling social contact and its contextual and affective features using experience sampling methodology) and these too offer a variety of methodological options for behavioral genetic research.

The inclusion of mutual friendships as part of the 'sibling effect' implies that sibling influence functions as part of a broader social context for smoking, a point emphasized by both Merikangas and Conger. Conger (2005) argues that there is a need to understand the larger social environment that includes parents as well as peers. Conger presents some of his seminal work demonstrating a variety of mechanisms by which parent, sibling and peer influences intersect. For example, a critical point for prevention is the finding that parent and sibling smoking may influence the acquisition of peers who smoke. Such directionality of effects suggests that family smoking may provide inroads to non-familial influences on smoking, a notion that is consistent with our model of sibling effects on smoking. The various levels of influence across social relationships discussed by Conger reinforces Merikangas'

point that social contextual effects reflect complex mechanisms which may be multiplicative rather than additive as well as her suggestion that the social contextual basis of adolescent smoking become a focus of prevention models.

In addition to this perspective on the social context, Brandon & Brandon (2005) offer an important emphasis on potential mechanisms by which social relationships transmit risk for smoking. Their focus on 'units of communication' reinforces the idea that social influences have impact via social cognitive processes, again a critical point for prevention studies. They offer an intriguing idea that sibling effects may operate in part by influencing the creation of expectancies about the consequences of smoking. This suggestion makes the broader point that work on social contextual influences on smoking may and should be linked with specific mechanisms that influence the individual.

In summary, the four commentaries offer a broad array of stimulating ideas on social influence, prevention, measurement, and methodology. What is impressive is the convergence from a number of perspectives on the importance of sibling influence on smoking. Sibling designs offer unique opportunities to examine familial and non-familial influences and provide opportunities to test both etiological and prevention models. Genetically informative sibling designs offer a further layer of potential by allowing for more insight into environmental factors as well as carry promise for eventually integrating both social and genetic influences on smoking.

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